

Dietary Fat Intake and Endothelial Dysfunction among Hypertensive Women

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## Abstract

High fat diets lead to low-grade systemic inflammation, a known cause of endothelial dysfunction and hypertension. Current research suggests that dietary modifications, including increasing unsaturated and lowering saturated fat intake, effectively treat hypertension.

Adherence to recommendations to promote heart health can be low and influenced by factors such as age. This study was designed to examine associations among dietary intake of heart-healthy (i.e., monounsaturated, polyunsaturated) and heart-unhealthy (i.e., saturated) fats and endothelial function in hypertensive women as well as the predictive value of age for dietary fat intake. This secondary analysis utilized data obtained from a pilot study designed to examine indicators of vascular function in chronic hypertension. A convenience sample of 11 women aged 30-60 years was enrolled. The Diet History Questionnaire II (DHQII) was used to measure typical fat intake over a 30-day period. Endothelial function was measured using an Endo-PAT (Itamar Medical; Israel) and based on reactive hyperemic index (RHI), with greater RHI reflective of better vascular function. Spearman rank-order correlations examined relationships among age, dietary fat intake, and endothelial function ( $\alpha = 0.05$ ). Mean dietary intake of monounsaturated, polyunsaturated, and saturated fats was 23.2 grams  $\pm$  12.2, 13.4 grams  $\pm$  9.0, and 18.7 grams  $\pm$  8.3, respectively. Monounsaturated ( $r_s = 0.50, p = 0.12$ ), polyunsaturated ( $r_s = 0.49, p = 0.13$ ), and saturated fat intake ( $r_s = 0.49, p = 0.13$ ) showed a trend toward improved vascular function, though no statistically significant association was identified. Greater age was associated with significantly less polyunsaturated ( $r_s = -0.60, p = 0.05$ ) and monounsaturated ( $r_s = -0.66, p = 0.03$ ) fat intake and marginally less saturated fat intake ( $r_s = -0.48, p = 0.14$ ) intake. This study advances understanding of the influence of dietary fat consumption on endothelial function, highlighting dietary modifications as a potential early intervention to improve

cardiovascular health. Dietary fat intake was not significantly associated with endothelial function, likely as a function of small sample size. The identified trends support the need for future studies examining the relationship between dietary fat intake and endothelial function, and the role of unmodifiable factors that may affect dietary patterns, such as age.

## **Chapter I: Statement of the Problem**

Hypertension is a manifestation of cardiovascular disease that contributes to cardiovascular-related morbidity and mortality affecting approximately 30% of American adults. Underlying hypertension is endothelial dysfunction, damage to the single layer lining of the vasculature that controls arterial tone (Dharmashankar, & Widlansky, 2010). In a healthy vessel, the endothelial cells contribute to the tension of the vascular smooth muscle via balanced release of vasodilating and constricting factors. Damage to the endothelium impairs the release of vasodilators and increases the release of vasoconstrictors, leading to decreased bioavailability of vasodilating factors and sustained constriction of the vessels, which in turn raises blood pressure and contributes to hypertension (Versari, Daghini, Virdis, Ghiadoni, & Taddei, 2009).

Treatment methods for hypertension can be approached both pharmacologically and non-pharmacologically. Non-pharmacological treatment includes lifestyle changes such as controlling weight, regular physical activity, smoking cessation, decreasing alcohol intake and maintaining a healthy diet. These changes have also been found to improve endothelial dysfunction. The American Heart Association recommends replacing saturated fats with unsaturated fats, specifically polyunsaturated fats, in order to decrease the incidence of cardiovascular disease (Sacks et al., 2017). Although non-pharmacologic methods are considered to be the first line of hypertensive treatment, only one in five patients diagnosed with hypertension in the United States adhere to dietary recommendations for management of the disease process (Kamran, Azadbakht, Sharifirad, Mahaki, Mohebi, 2015). Perceived barriers to lifestyle changes such as food preferences, financial limitations, time restraints, and lack of social support severely hinder self-efficacy of patients diagnosed with cardiovascular diseases

(Krummel, Humphries, Tessaro, 2002) and serve as the main predictor of health-promoting behaviors (Kamran et al., 2015).

### *Background of the Problem*

Hypertension develops as a direct result of inflammation of the vasculature in conjunction with oxidative stress; decreasing the prevalence of these two factors in the system has been shown to improve endothelial function (Dharmashankar, & Widlansky, 2010). Oxidative stress leads to the release of free radicals, which antagonize vasodilators and cause vessels to contract. Endothelial dysfunction occurs as a result of this excessive vasoconstriction. Diets high in saturated fat lead to inhibition of high density lipoprotein (HDL) anti-inflammatory and antioxidant effects (Barter, Nicholls, Anantharamaiah, Navab, & Fogelman, 2004), as well as absorption of bacteria by the gastrointestinal system causing low-grade systemic inflammation and perpetuating oxidative stress (Ruiz-Nuñez, Dijck-Brouwer, Muskiet, 2016).

Current research was analyzed by the American Heart Association to create dietary recommendations and found that replacing saturated fat with polyunsaturated or monounsaturated fat in the diet of healthy individuals yields increased HDL levels, yielding increased protective properties within the vasculature (Mensick, 2016). Further, increasing intake of polyunsaturated and monounsaturated fats improved vascular reactivity and a single meal in healthy adults (Hall, 2009). These studies show the effects of interventions to alter saturated fat intake, but are limited by investigating participants with no known cardiovascular risk factors. This leads to a lack of information on the relationships between dietary fat intake of individuals diagnosed with hypertension.

### *Purpose of the Study*

According to the American Heart Association Presidential Advisory on Dietary Fats and Cardiovascular disease (Sacks et al., 2017), the use of polyunsaturated fats, monounsaturated fats, and carbohydrates in place of saturated fats in the diet decrease incidence of cardiovascular disease. These recommendations stem from research studies which manipulated saturated fat intake in healthy individuals to analyze the effects on components of vascular reactivity. This study examines associations among monounsaturated fat intake, polyunsaturated fat intake, and saturated fat intake, and endothelial function in women undergoing current pharmacological treatment for chronic hypertension. Associations among age and dietary fat intake were also examined for congruity with American Heart Association recommendations. By analyzing relationships among dietary fat intake, endothelial function, and age, we hope to identify the role of dietary fat intake in endothelial dysfunction, informing dietary recommendations for women with hypertension.

### *Significance of the Study*

The American Heart Association recommends non-pharmacologic approaches to cardiovascular disease management by replacing saturated fat in the diet with polyunsaturated fats. This study will advance understanding of the influence of dietary saturated, monounsaturated, and polyunsaturated fat consumption which can be adapted as an early intervention to prevent the progression of endothelial dysfunction among women with hypertension and, by extension, cardiovascular disease.

### *Conceptual Frame of Reference (Theory)*

Pender's Health Promotion Model served as the framework of reference for this study. Pender's model allows for analysis of major determinants of health behaviors for development of intervention based upon the theory that increased awareness of health-promoting behaviors

yields positive health outcomes (Pender, 2011). Application of the theory provides information for detecting key aspects of health promotion behaviors among different age groups, genders, income, and education levels (Heydari, & Khorashadizadeh, 2014).

#### *Aims/Research questions*

This study will examine associations among dietary fat intake, endothelial function, and age in female hypertensive patients. Specifically, the following questions will be addressed: (1) In hypertensive women, does a diet containing heart-healthy (i.e. monounsaturated, polyunsaturated) fat or heart-unhealthy (i.e. saturated) fat intake associated with endothelial function? (2) In hypertensive women, how does age relate to dietary intake of fat?

#### *Definition of Terms*

The operational definitions of the following terms are defined for the purpose of this study. Hypertension is defined as a systolic blood pressure of 140 mmHg or higher or diastolic blood pressure of 90 mmHg or higher. Endothelial function is defined by the reactive hyperemia index (RHI) as measured by EndoPat-2000 (Itamar Medical; Israel). RHI values  $<1.67$  demonstrate vascular dysfunction (Bonetti et al., 2004). We define heart-healthy fats to include monounsaturated and polyunsaturated fats, and heart-unhealthy fats to include saturated fats. Recommended monounsaturated fat intake is defined as 10-15% of energy intake or 22-33 grams per day (g/day). Recommended polyunsaturated fat intake is defined as 7-10% of energy intake or 13-22 g/day. Recommended saturated fat intake is defined as 5-7% of energy intake, or 11-13 g/day (Sacks et al., 2017). All daily recommendations are based on a 2,000 calorie diet.

## **Chapter II: Review of Literature**

In order to evaluate the evidence for endothelial function, dietary fat intake and diet compliance, a review of literature was conducted. This chapter reviews the available evidence for (1) effects of oxidative stress and inflammation on vascular function, (2) effects of dietary interventions on oxidative stress inflammation, (3) dietary intervention compliance and vascular function, and (4) dietary intervention compliance and age.

### *Effects of Oxidative Stress and Inflammation on Vascular Function*

Hypertension occurs in the presence of oxidative stress, local inflammation, and systemic inflammation, but is largely asymptomatic prior the presentation of complications (Dharmashankar, & Widlansky, 2010). The risk and pathophysiology of hypertension is influenced by sympathetic nervous system hyperactivity, renin-angiotensin-aldosterone system (RAAS) stimulation, and endothelial dysfunction (Rahimmanesh, Shahrezaei, & Bahman, 2012). Stimulation of the sympathetic nervous system contributes to hypertension with the release of norepinephrine, which has vasoconstrictive properties. RAAS functions to increase blood volume and blood pressure. Angiotensin converting enzyme (ACE) converts angiotensin I to angiotensin II, a potent arterial vasoconstrictor. Angiotensin II stimulates the production of aldosterone in the adrenal cortex, leading to the reabsorption of sodium and water, further increasing blood pressure. Angiotensin II is also known to have a pro-inflammatory effect on leukocytes, vascular smooth-muscle cells, and endothelial cells (Rahimmanesh et al., 2012). Chronic-inflammation, in turn, has a deleterious effect on endothelial function. Endothelial dysfunction results in an imbalance of vasodilating and vasoconstricting factors, contributing to sustained vasoconstriction. An increase in adhesion to the vessel walls is also observed, decreasing available volume for blood flow and increasing pressure.



The endothelium, when functioning correctly, is protective by inhibiting platelet, leukocyte, and monocyte adhesion, as well as proliferation of smooth muscle cells, and maintenance of physiologic permeability (Boulanger, 1999). According to Flammer et al. (2012), endothelial dysfunction is defined as a “pathological condition characterized mainly by an imbalance between substances with vasodilation, antimitogenic, and antithrombogenic properties and substances with vasoconstricting, prothrombotic, and proliferative characteristics” (p. 753). Endothelium-derived relaxing factors (EDRFs) are inhibited in endothelial dysfunction, leading to excessive vasoconstriction. Nitric oxide (NO), a potent vasodilator and EDRF, inhibits platelet aggregation, platelet adhesion, leukocyte migration, and smooth muscle cell proliferation (Flammer et al., 2012). Angiotensin II, endothelins, and NO antagonists, such as reactive oxygen species (ROS) cause contraction of the dysfunctional vessels (Rahimmanesh et al., 2012). The combination of EDRF breakdown, overproduction of contracting factors, and oxidative stress create endothelial dysfunction.

The interactions between endothelial dysfunction and hypertension must be considered in order to fully understand the pathophysiology. Hypertension is believed to develop as a direct result of inflammation of the vasculature in conjunction with oxidative stress. Yet, both hypertension and endothelial dysfunction have been shown to improve with decrease in the prevalence of these two factors in the system (Dharmashankar, & Widlansky, 2010). Hypertension can present with varying degrees of endothelial dysfunction. The most common presentation of endothelial dysfunction is identified by low bioavailable nitric oxide due to breakdown by ROS, which are products of increased oxidative stress (Versari et al., 2009).

Endothelial dysfunction and hypertension both share the pathophysiology of oxidative stress and vascular inflammation, partially explaining their interactions and common

simultaneous presentation. The relationship between these disease processes is considered bidirectional. It has been found that risk of hypertension is increased six-fold in normotensive women with impaired endothelial function (Dharmashankar & Widlansky, 2010).

Cardiovascular risk is increased in those with endothelial dysfunction, which is furthered by the presence of other cardiovascular risk factors. The correlating factors between hypertension and endothelial dysfunction indicate that treatments aimed at reversing either condition may help resolve the other.

### *Effects of Dietary Interventions on Oxidative Stress and Inflammation*

The American Heart Association recommends decreasing dietary saturated fat intake as a means of intervention for both hypertension and endothelial dysfunction (Sacks et al., 2017).

High-fat diets that replace saturated fat intake with polyunsaturated or monounsaturated fat have been found to increase HDL levels. This increase is even higher when saturated fats are replaced by carbohydrates in the diet (Mensick, 2016). HDL serves the primary function of removing cholesterol from arterial walls; however, HDL also has an antioxidant and anti-inflammatory effect on the blood vessels (Barter, Nicholls, Anantharamaiah, Navab, & Fogelman, 2004).

Studies have shown that diets high in saturated fat yield impaired vascular reactivity, with a single meal high in saturated fat negatively impacting the ability of HDL to protect the endothelium. These effects were mediated by meals high in polyunsaturated and monounsaturated fats, which increased vascular blood flow (Nicholls et al., 2006). Hall (2009) conducted a literature review of data surrounding saturated fat intake, unsaturated fat intake and vascular function and found that not only did diets high in unsaturated fats exhibit protective properties against cardiovascular disease, but when unsaturated fat intake was increased to replace saturated fat intake, vascular reactivity improved.

*Dietary Intervention Compliance and Vascular Function*

The prevalence and implications of endothelial dysfunction necessitates interventions in order to decrease the risk of cardiovascular morbidity and mortality. Treatment of endothelial dysfunction is approached with the intention of reinstating endothelium dependent vasodilation and increasing NO levels (Rahimmanesh et al., 2012). The American Heart Association recommends the use of heart healthy diets such as the DASH diet, which has been shown to prevent and manage high blood pressure, while also improving endothelial dysfunction (Kim, 2016). Lifestyle changes have the ability to improve, if not reverse, hypertension and endothelial dysfunction, either with or without concurrent pharmacological treatment.

A cross-over study by Swain et al. (2012) examined the macronutrient proportions in various heart-healthy diets while maintaining 6% saturated fat intake, and 100-200 mg of cholesterol per day. Three structured diets were tested; a carbohydrate-rich diet, a diet which replaced 10% of carbohydrates with protein, and a diet which replaced the same 10% of carbohydrates with unsaturated fats. The study found a significant reduction in cardiovascular risk factors such as blood pressure, serum low density lipoproteins (LDL), serum HDL, and triglycerides for all three diets. This reduction in risk was even greater in the protein and unsaturated fat-rich diets. The study provides evidence of the correlation between saturated fat intake and cardiovascular risk.

DASH diet compliance is believed to improve endothelial function, a theory supported by the findings of Rifai et al. (2015) cluster study comparing participation in the DASH diet and endothelial function, as measured by large artery elasticity over a three-month period. The study concluded that large artery elasticity improved in the first month but slowed in the latter two months of the study. This indicates a decrease in the progression of endothelial dysfunction in

these hypertensive patients. The results also showed an increased compliance to the dietary recommendations over time, suggesting a longer, longitudinal study may be able to witness further improvement in endothelial function.

The DASH diet recommends an intake rich in fruits, vegetables, protein, and low-fat dairy products, paired with reduced saturated fat and total fat intake (Swain et al, 2012). Per a secondary analysis by Kim (2016), compliance with the DASH diet is often low, especially in those with a diagnosis of hypertension. This analysis also found that the non-compliant diets of the test population were higher in sodium, saturated fat, total fat and protein intake than those without a diagnosis of hypertension. Most of these dietary choices have been shown to exacerbate hypertension. Lower sodium intake has demonstrated a mild antihypertensive effect and can improve endothelial dysfunction related to sodium dietary intake or secondary to hypertension (Dharmashankar & Widlansky, 2010) while saturated fat intake has been linked to higher cardiovascular risk (Swain et al., 2012).

#### *Dietary Intervention Compliance and Age*

The success of lifestyle changes as treatment options for conditions such as hypertension and endothelial dysfunction are heavily dependent on patient compliance. Interestingly, while patients with chronic conditions of advanced age are found to have higher adherence rates to pharmacological management methods of disease management (Alefishat, Farha, & Al-Debei, 2016), it is estimated that only 20% of patients diagnosed with hypertension follow dietary recommendations to reduce salt intake alone (Kamran, Azadbakht, Sharifirad, Mahaki, & Mohebi, 2015). Individual health promotion behaviors are influenced by individual factors such as self-efficacy, perceived benefits and barriers, as well as interpersonal and social factors (Heydari, & Khorashadizadeh, 2014). Within these, self-efficacy functions as a predictor of fat

and fiber intake (Krummel, Humpries, & Tessaro, 2002). According to Kamran et. al. (2015), perceived barriers, such as comorbidities, poor health status, and limitations to adopting lifestyle changes, were found to have the most influence on patient behavior. The perception of these barriers increased with age (Kamran et al, 2015).

### **Chapter III: Methodology**

This secondary analysis was developed based on the primary study, designed to examine the potential correlation of future risk for cardiovascular disease after preeclampsia with DNA methylation in cardiovascular-related genes and vascular function. Data for behavioral variables including diet and activity were collected. The purpose of this chapter is to describe the methodology of this secondary analysis designed to identify the role of dietary fat intake in endothelial dysfunction in order to inform dietary recommendations for women with hypertension, to include the research design, population and sample design, data collection procedures, and data collection instruments, and protection of human subjects.

#### *Research Design*

The parent study, a pilot study, used convenience sampling and an observational design. This study is a cross-sectional secondary analysis utilizing data obtained by questionnaires and chart review to examine the relationships among dietary fat intake, endothelial function, and age.

#### *Population and Sample Design*

In the parent study, potential participants were recruited from a population in the upper Midwest. Advertisements describing the study were placed in clinic waiting areas where participants were likely to be. Human subjects' protection was assured throughout the study. The Institutional Review Boards of the University of North Dakota and Altru Health System approved the study. Eligibility criteria included English-speaking women, age 30-60, with a diagnosis of chronic hypertension undergoing current treatment, and a history of at least one prior pregnancy. Exclusion criteria included current pregnancy, and presence of comorbid condition(s) that influence cardiovascular health (e.g. autoimmune, diabetes, congenital cardiac anomalies).

Potential participants meeting inclusion criteria attended a single study visit where eligibility was reconfirmed. Participants were verbally informed of the expectations of participation, the risks, the discomforts, potential adverse reactions, their right to withdraw from the study at any time, as well as methods to promote confidentiality. This information was also provided in writing and time was provided for participants to read this over and ask questions. At the conclusion, if they wished to participate, they signed a written consent. A copy was provided to the participant and the originals were retained in a secure location.

#### *Data Collection Procedures*

All data were collected during the single visit in a private space. After obtaining the informed consent, data were collected to include medical history, demographic information, and physical activity report by a questionnaire. The Diet History Questionnaire (DHQII, Version 2.0) was also completed and collected electronically to provide data on dietary intake at this time. Blood pressure was measured using standard procedures and vascular function was measured using the Endo-PAT2000 (Itamar Medical; Israel). Medical record data were retrieved from the investigator.

#### *Data Collection Instruments*

Data for dietary intake was based on the Diet History Questionnaire (DHQII, Version 2.0), a food frequency questionnaire developed by staff at the Risk Factor Assessment Branch (RFAB) of the National Institute of Health (NIH). The DHQII is comprised of 134 food items including portion size and 8 dietary supplement questions. It required participants to select food which represented their dietary and supplement intake over the previous 30 days. Self-reported dietary fat intake estimated as grams per day (g/day) were used for analyses. Total saturated fat

intake was included as a measure of heart-unhealthy fat intake. Total monounsaturated fat intake and total polyunsaturated fat intake were included as measures of heart-healthy fat intake.

Vascular function data were collected using an Endo-PAT2000. Peripheral Arterial Tone (PAT) technology measured changes in arterial pulsatile blood volume at the fingertip related to sympathetic activation in both a control and occluded arm. Vascular function was measured in a quiet location and all jewelry and watches were removed from the participants' arms. Blood pressure was recorded after the participant was placed in the supine position 5 minutes before vascular function was tested. A baseline vascular function reading was taken in both arms for 5-10 minutes (baseline period), followed by a 5-minute period of occlusion of one arm via blood pressure cuff (occlusion period). The pulsatile blood volume was recorded for 5 minutes following the deflation of the cuff (test period). This was recorded on a graph in which the amplitude of the measurements indicated the level of arterial pulsatile blood volume.

The technology provided values for the calculation of a RHI (i.e., the post-to-pre-occlusion PAT-Ratio (test period/baseline period) in the occluded arm compared to the same in the control arm). An RHI value  $<1.67$  indicates endothelial dysfunction and cardiovascular impairment (Bonetti et al., 2004).

### *Analysis*

Endothelial function data were available for 11 of the 12 women with corresponding intake data. Thus, we analyzed 11 women with data for both measures. Descriptive statistics including count [frequency] and mean [standard deviation] were used to describe the sample. Spearman rank-order associations were examined to identify relationships among dietary intake of saturated fat, polyunsaturated fat, and monounsaturated fat, endothelial function, and age. Statistical significance was defined according to  $\alpha = 0.05$ .



*Protection of Human Subjects*

All data sheets and response forms were coded with a unique number in order to protect the human subjects' identity and information. All involved staff and volunteers involved in this study completed education on the protection of human subjects. The study was approved by the IRBs at the University of North Dakota and Altru Health System.

**Chapter IV: Results:***Sample Descriptive Statistics:*

In this secondary analysis, we analyzed a sample used in the primary study which included 11 English-speaking women, age 35-60, with a history of at least one prior pregnancy, and a diagnosis of chronic hypertension undergoing current treatment. As shown in Table 1, the average age of participants was 53 years, and 73% are current or former smokers. All participants were in the overweight or obese BMI category, with an average BMI of 35.2. The average reactive hyperemic index (RHI) was 1.6, with 36% ( $n = 4$ ) of patients with RHI in the healthy range ( $> 1.67$ ). The mean average monounsaturated and polyunsaturated intakes for participants fell within American Heart Association recommendation parameters, however, the mean saturated fat intake of participants fell 5.7 g/day or 43.8% above the recommended intake.

**Table 1. Descriptive Statistics**

<b>Table 1. Sample Descriptive Statistics (n=11)</b>	
Age (Mean, (SD))	53 (8)
Education (Count, (Frequency))	
$\leq$ Some college	5 (45%)
$\geq$ Bachelor's degree	6 (55%)
Income (Count, (Frequency))	
$<$ \$20,000/year	5 (45%)
\$20,000 - \$34,999/year	1 (10%)
$\geq$ \$35,000/year	5 (45%)
Walking Frequency (Count, (Frequency))	
0 days per week	4 (37%)
1-3 days per week	2(18%)
4-7 days per week	5 (45%)
Smoking Frequency (Count, (Frequency))	
Never smoker	3 (27%)
Former smoker	6 (55%)
Current smoker	2 (18%)
BMI (Mean, (SD))	35.2 (11.7)
Reactive Hyperemia Index (Mean, (SD))	1.6 (0.5)
Monounsaturated Fat Intake [grams/day] (Mean, (SD))	23.2 (12.2)
Polyunsaturated Fat Intake [grams/day] (Mean, (SD))	13.4 (9.0)
Saturated Fat Intake [grams/day] (Mean, (SD))	18.7 (8.3)

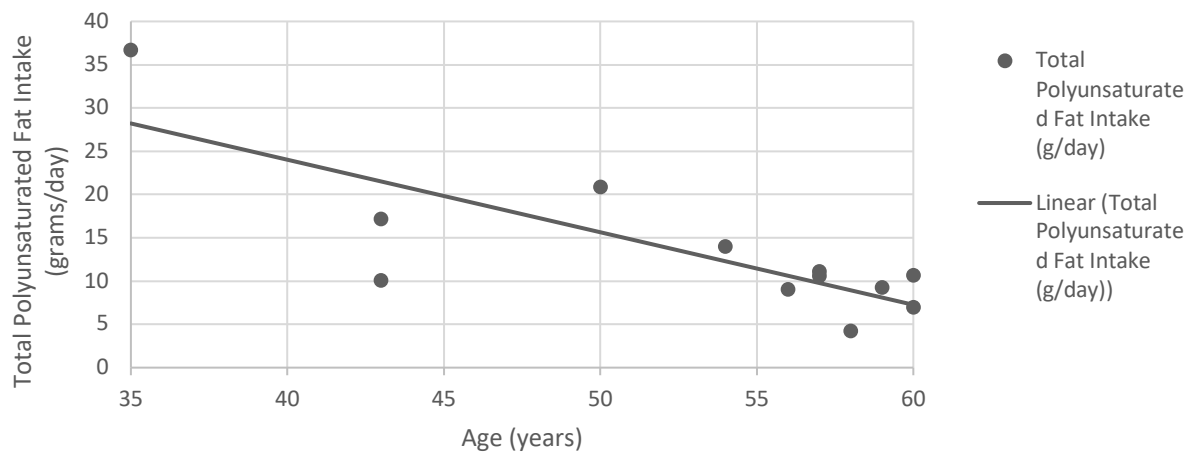
Age and Dietary Fat Intake

Associations between dietary polyunsaturated, monounsaturated, and saturated fat intake, age, and RHI were examined using Spearman associations as shown in Table 2. Greater age was associated with significantly less polyunsaturated ( $r_s = -0.60$ ,  $p = 0.05$ ; Figure 1) and monounsaturated ( $r_s = -0.66$ ,  $p = 0.03$ ; Figure 2) fat intake. Greater age was associated with marginally less saturated ( $r_s = -0.48$ ,  $p = 0.14$ ; Figure 3) fat intake. No other associations were significant.

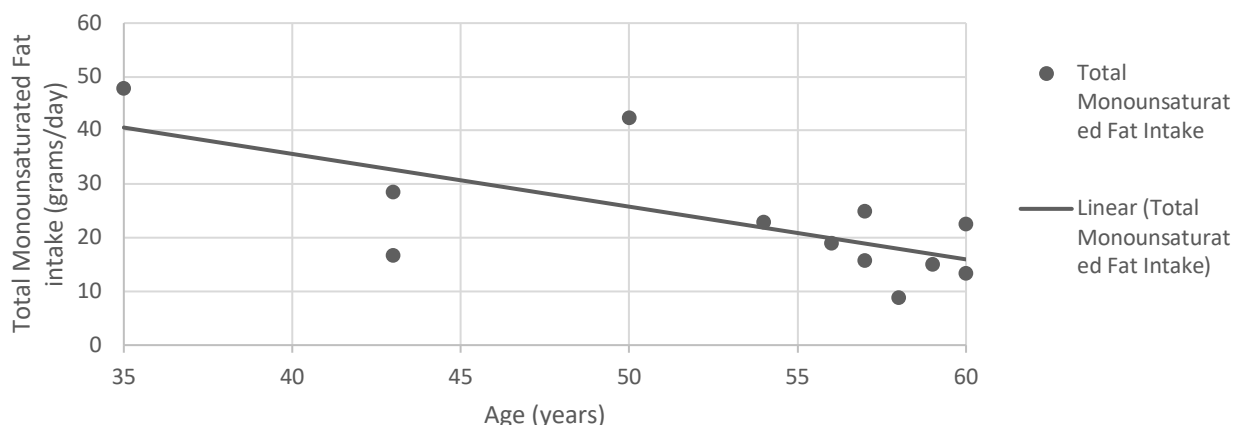
Table 2. Correlation Table: Age and Fat Intake

Table 2. Correlation Matrix: Age and Fat Intake				
	Age	Saturated Fat Intake	Monounsaturated Fat Intake	Polyunsaturated Fat Intake
Age	1.00	--	--	--
Saturated Fat Intake	-0.48	1.00	--	--
	0.14			
Monounsaturated Fat Intake	-0.66	0.90	1.00	--
	0.03	0.00		
Polyunsaturated Fat Intake	-0.60	0.93	0.94	1.00
	0.05	0.00	0.00	

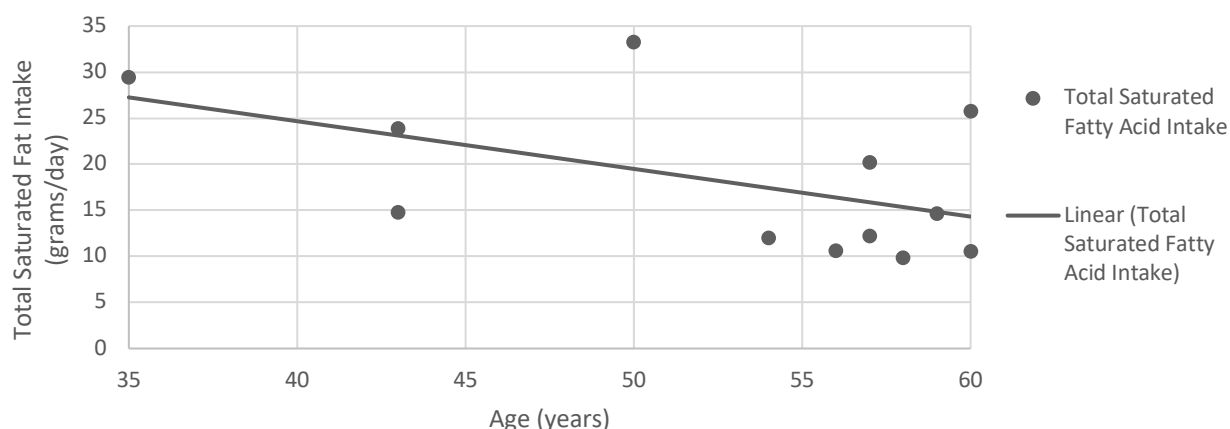
Figure 1. Age and Total Polyunsaturated Fat Intake



**Figure 2. Age and Total Monounsaturated Fat Intake**



**Figure 3. Age and Total Saturated Fat Intake**



### *RHI and Dietary Fat Intake*

Spearman rank-order associations among dietary fat intake and RHI are shown in Table 3. Polyunsaturated fat intake, monounsaturated fat intake, and saturated fat intake were each positively but non-significantly associated with RHI ( $r_s = 0.49$ ,  $p = 0.13$ , Figure 4;  $r_s = 0.50$ ,  $p = 0.12$ , Figure 5; and  $r_s = 0.49$ ,  $p = 0.13$ , Figure 6, respectively). These findings showed a trend toward improved endothelial function with greater dietary fat intake.

**Table 3. Dietary Fat Intake and RHI**

Table 3. Correlation Matrix: RHI and Fat Intake				
	Saturated Fat Intake	Monounsaturated Fat Intake	Polyunsaturated Fat Intake	RHI
Saturated Fat Intake	1.00	--	--	--
Monounsaturated Fat Intake	0.90	1.00	--	--
Polyunsaturated Fat Intake	0.93	0.94	1.00	--
RHI	0.49	0.50	0.49	1.00
	0.00	0.00	0.13	

Figure 4. Total Polyunsaturated Fat Intake and RHI

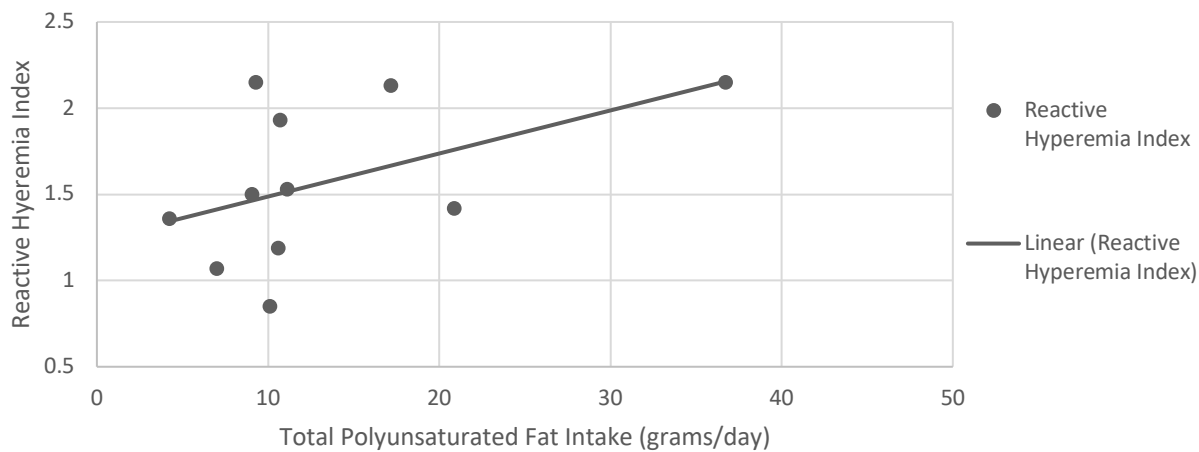


Figure 5. Total Monounsaturated Fat Intake and RHI

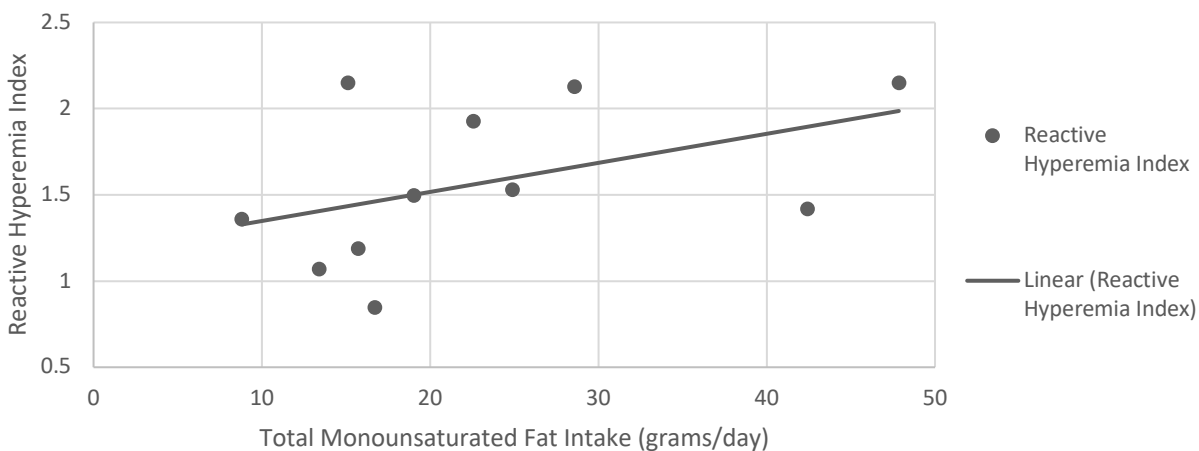
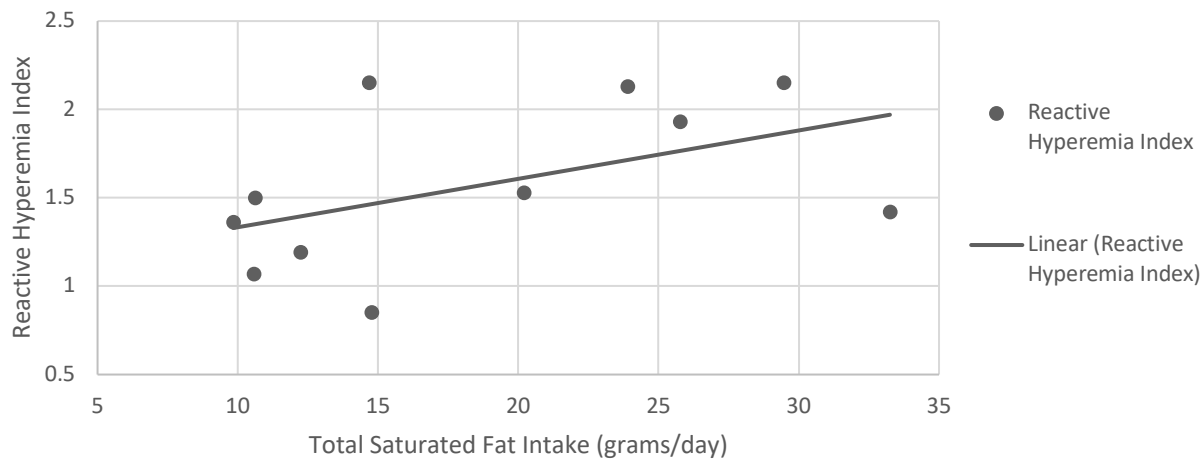


Figure 6. Total Saturated Fat Intake and RHI

Running Head: Dietary Fat Intake in Hypertensive Women



## **Chapter V: Discussion**

### *Summary of Findings*

This study purpose was to identify the role of dietary fat intake in endothelial dysfunction in order to inform dietary recommendations for women with hypertension. We found that women of greater age reported marginally lower saturated fat intake, this was paralleled by significantly lower monounsaturated and polyunsaturated fat intake. Greater monounsaturated, polyunsaturated, and saturated fat intakes were positively but non-significantly associated with better endothelial function.

The associations between increased age and decreased dietary fat intakes were unexpected, as literature supports a decrease in dietary compliance with increased age and in the presence of hypertension diagnosis (Kim, 2016). The relationship between intake of polyunsaturated, monounsaturated, and saturated fat suggests the potential for a decrease in all fat intake as motivated by increased awareness. This is supported by Pender's health promotion model framework, which posits that patient education may yield increase in patient perception of self-efficacy, benefits of health-promoting behaviors, and control of health. It is also notable that lower levels of saturated fat intake were paralleled by lower levels of unsaturated fat intake, which does not conform to American Heart Association recommendations to shift fat intake away from heart-unhealthy fats (i.e., saturated) and toward heart-healthy fats (i.e., unsaturated). Additional education in this area may be warranted, particularly among women being treated for hypertension. This decrease in intake could also be related to a decrease in overall consumption, which was not analyzed by this study.

In addition, greater monounsaturated and polyunsaturated fat intakes were associated with a trend toward improved endothelial function. This was consistent with the literature found,

which displayed improved vascular reactivity and blood flow with increased unsaturated fat intakes (Hall (2009). The protective effects of these dietary fats may explain the concurrent finding of better endothelial function with increased saturated fat intake, as unsaturated and saturated fat intakes were highly associated among our participants. This covariation may have masked any detrimental effects of the saturated fat in participants' diets.

The trends and associations of these results suggest that future studies in a larger, more ethnically diverse population could potentially identify further significance between age and dietary recommendation compliance, and between dietary fat intake and endothelial function.

### *Limitations*

Limitations of this study include a small and non-diverse sample of participants, which limits generalizability. Specifically, exclusion criteria for participants included current pregnancy and presence of comorbid condition(s) that influence cardiovascular health (e.g. autoimmune, diabetes, congenital cardiac anomalies). Additionally, the study's sample included all Caucasian women.

### *Recommendations*

A greater understanding of the relationships between dietary fat intake, endothelial health, and age can aid in designing and delivering health-promoting dietary interventions for hypertensive patients who may or may not be experiencing endothelial dysfunction. Further investigation is needed to determine the exact motivations of interventions and education to encourage patient participation and understanding. Further research with larger sample sizes and longitudinal designs would allow for analysis of causative factors in endothelial dysfunction in relation to dietary fat intake in patients undergoing treatment. This would contribute to existing



evidence as to the importance of dietary changes in the presence of endothelial dysfunction and hypertension.

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